Dissecting aneurysms of the intracranial arteries are exceedingly rare vascular lesions that produce acute cerebral or brain-stem infarction in young, otherwise healthy adults. Unlike dissections of the cervical carotid arteries, which usually resolve spontaneously, intracranial dissecting aneurysms carry a high mortality rate. These lesions often simulate cerebral thrombosis and can be overlooked in the evaluation of a patient with cerebral vascular insufficiency of sudden onset, thus, their true incidence has been underestimated. In this report, we present six cases of dissecting aneurysm of the posterior circulation, and a review of the literature relating to this disorder. The pathology, clinical presentation, angiographic findings, and surgical management are also discussed.

Case Reports

Case 1

This 46-year-old man began to have intermittent headaches on the right side 9 days before admission. The pain intensified after onset and was accompanied by a stiff neck, fever, and chills. Lumbar puncture yielded bloody cerebrospinal fluid (CSF). The patient had no history of hypertension, diabetes, smoking, or trauma. The neurological findings during his initial evaluation at another hospital were normal. A computerized tomography (CT) scan showed an enhancing mass on the right in the posterior fossa. Vertebral angiography revealed a partially thrombosed 1.5 x 2-cm aneurysm at the right verteobasilar junction (Fig. 1). The headaches resolved within 4 days and the patient was transferred to the University of California, San Francisco (UCSF), for further evaluation.

Operation. Three days after admission, the patient underwent a right suboccipital craniectomy. The leptomeninges were yellow, which indicated prior subarachnoid hemorrhage (SAH). The distal 2 to 3 mm of the right vertebral artery near its basilar junction were normal, but the proximal segment expanded into a...
Dissecting aneurysms of the posterior circulation

FIG. 1. Case 1. Right vertebral angiograms, anteroposterior (left) and lateral (right) views, showing an irregularly shaped, partially thrombosed dissecting aneurysm at the right vertebrobasilar junction (arrows).

tubular, purple-red mass ventral to the ninth and 10th cranial nerves, indenting the medulla. The dilated segment had an irregular surface and was approximately 1.5 cm in diameter. The right PICA was expanded at its junction with the vertebral artery. One clip was placed across the proximal PICA near its junction with the vertebral artery, and another clip was placed proximal to the vertebrobasilar junction. The vertebral artery was ligated extracranially. Needle puncture of this isolated sac produced a small stream of bright red blood. No further attempt was made to find the source of residual flow.

Postoperative Course. The patient was alert, but within 48 hours after surgery he deteriorated neurologically and required reintubation. Clinical signs of pontomedullary infarction were present. A CT scan revealed a hemorrhagic right cerebellar infarction and ventricular enlargement. After placement of a ventricular catheter to drain CSF, he began to follow commands and regained motor control on the left side. Neurological deterioration continued, and the pattern of progression indicated a basilar artery thrombosis. His hospital course was complicated by intermittent aspiration pneumonitis, sepsis, and thrombocytopenia. Eight weeks after operation, he had made a nearly complete recovery from his neurological deficits, although he still had difficulty with swallowing, and was transferred to a rehabilitation facility. He did well for 6 months, but died of non-neurological causes (bleeding diathesis).

Case 2

This 39-year-old man presented to a local emergency room for treatment after the abrupt onset of severe occipital headache and neck stiffness. Lumbar puncture yielded bloody CSF, but a CT scan was negative. Four-vessel angiography showed a fusiform dilation of the proximal basilar artery, and he was transferred to UCSF.

Examination. The patient was confused, but had no other neurological symptoms. Repeat angiography (Fig. 2) showed that the left vertebral artery was irregular distal to the origin of the PICA. In the proximal basilar artery, there were several relatively stenotic segments with intervening areas of irregular vessel caliber consistent with vasospasm. Reflux into the contralateral vertebral artery demonstrated a segment of focal enlargement at the origin of the PICA.

First Operation. One week after admission to the hospital, the patient underwent a right suboccipital craniectomy. Surgical exploration disclosed evidence of prior SAH and fusiform enlargement of the right vertebral artery beginning at its entry into the subarachnoid space. The vessel was discolored and had a neovascular pattern in its thin outer wall; these findings suggested a diagnosis of dissecting aneurysm. The upper extent of the lesion could not be identified and nothing further was done. Ventriculomegaly developed postoperatively and resulted in abrupt clinical deterioration. A ventricular catheter was placed for CSF drainage. The patient responded well, but developed a Staphylococcus aureus CSF infection. The infection was adequately treated and eventually a ventriculoperitoneal shunt was inserted. He was discharged in good condition, but required a shunt revision 3 months later.

Eleven months after the initial attack, the patient was readmitted with dizziness and diplopia. Neurological assessment revealed bilateral horizontal and vertical
M. S. Berger and C. B. Wilson

Second Operation. Exploration of the left vertebral artery disclosed an aneurysm, without evidence of dissection. The neck of the aneurysm was large and firm and would not accommodate a clip placed across it. The base of the aneurysm was packed with muslin strips after it developed a small rent. Postoperatively, the patient had mild dysphagia and hoarseness, but was discharged in stable condition.

Third Operation. Progressive brain-stem compression developed over the next several weeks; therefore, the proximal left vertebral artery was occluded with two detachable balloons just above the origin of the subclavian artery. The distal basilar artery was filled adequately by the anterior circulation. A CT scan 1 week later showed partial thrombosis of the aneurysm. Repeat angiography 3 months later demonstrated residual filling of the lumen of the aneurysm from muscular branches of the ascending cervical and occipital arteries.

Fourth Operation. The patient underwent reexploration, thrombectomy, and partial aneurysmectomy. A large clip was placed inferomedially, sparing the PICA, which originated from the most caudal part of the aneurysm’s neck. A postoperative angiogram showed minimal filling of the base of the aneurysm and filling of the PICA. Four months later, the patient’s condition is stable although he has persistent dysphagia, hoarseness, mild ataxia, and hypesthesia on the left side of the face.

Case 3

This 43-year-old man was well until he began to have occipital headaches. Three weeks later, after lifting weights, he noticed the onset of double vision on lateral gaze and had difficulty with balance. Forty-eight hours later he began to have difficulty adducting the right eye. A CT scan revealed an enhancing lesion in the region of the distal basilar artery, and the patient was transferred to UCSF for further evaluation. He had no
Dissecting aneurysms of the posterior circulation

FIG. 4. Case 3. Anteroposterior (left) and lateral (right) views of the left vertebral angiograms showing diffuse fusiform dilation of the distal basilar artery and both proximal posterior cerebral arteries (arrowheads). The dissection also involves the right superior cerebellar artery (arrows).

history of hypertension, but had a 25-pack/year history of smoking.

Examination. Neurological assessment revealed right internuclear ophthalmoplegia, sixth nerve paresis, nystagmus in all directions of gaze, dysarthria, and dysmetria on the left side. Lumbar puncture revealed no blood in the CSF. A left vertebral angiogram (Fig. 4) revealed a fusiform dilation of the basilar tip involving both proximal (P\textsubscript{1} segments) of the posterior cerebral arteries (PCA’s) and the superior cerebellar artery on the right. The basilar artery proximal to the dilated segment was narrowed. The patient’s symptoms slowly resolved, and a repeat angiogram 2 months later showed that the P\textsubscript{2} segment on the right was involved (Fig. 5). A follow-up angiogram 1 month later showed no further progression of the disease. These clinical and angiographic features suggested a diagnosis of dissecting aneurysm of the aforementioned vessels.

Operation. Surgical exploration was performed through a right frontotemporal craniotomy, and the exposed enlarged vessels of the distal basilar trunk were reinforced with muslin strips. The outer wall of the dilated basilar tip was dull white and did not pulsate; beneath it, there was serous fluid, which represented resolution of the intramural hematoma. The small perforating arteries from the P\textsubscript{1} segments bilaterally were not involved in the dissection.

Postoperative Course. The patient’s postoperative course was uneventful and he was discharged 8 days after surgery. Three months later, the occipital headaches returned. A repeat vertebral angiogram showed complete occlusion of the right PCA distal to its junction with the posterior communicating artery. Aspirin therapy was started for suspected thrombosis, and his condition remains stable 5 months later.

Case 4

This 21-year-old woman was well until she began to have severe bilateral frontotemporal headaches. Lumbar puncture yielded slightly xanthochromic CSF, which was attributed to SAH. A CT scan showed an enhancing lesion in the distribution of the distal left PCA. The angiogram showed a fusiform dilation of the temporal branch of the left PCA (Fig. 6). The patient had a history of cardiac murmur, use of oral contra-
ceptive agents, and occasional use of oral amphet-
amines.

Examination. The results of her neurological examination on admission to UCSF were normal. Physical examination confirmed a Grade II/VI systolic ejection murmur; the electrocardiogram and echocardio-
gram were normal. She was afebrile, and evaluation for infective endocarditis was negative.

Operation. Three days after admission, the patient underwent a left frontotemporal craniotomy. Approxi-
mately 1.5 cm from the origin of the P2 segment, there was a large dilated branch vessel attached to the under-
surface of the temporal lobe. The mass was brown and firm, 3 cm long, and 1.5 cm in diameter. These findings were consistent with a diagnosis of dissecting aneurysm. The abnormal segment was clipped on both sides and resected. Pathological examination revealed a throm-
bosed, partially organized arterial segment. The internal elastic lamina and media were completely replaced by connective tissue, which formed the periphery of the intramural thrombus. No inflammatory cells were seen and tissue culture was negative for organisms. The adventitia was intact.

Postoperative Course. The patient had mild right hemiparesis and a transient movement disorder of the right upper extremity. Her condition is stable 48 months after resection of the dissecting aneurysm.

Case 5

This 57-year-old man suffered the abrupt onset of diffuse headache and diplopia and went to the local emergency room. The patient had no history of hyper-
tension, but had an 80-pack/year history of smoking. Bilateral sixth nerve paresis was present. Lumbar puncture yielded bloody CSF. A CT scan was negative, but left vertebral angiography showed a fusiform dilation at the tip of the basilar artery. Four weeks later, dysarthria and a mild right hemiparesis developed, and he was transferred to UCSF. Repeat angiography showed no change from the previous study. Surgical exploration of the basilar artery revealed an atherosclerotic dilation of the distal end, which appeared to be the origin of the SAH. The pathological findings were thought to be consistent with a dissecting aneurysm that had bled. The exposed abnormal portions of the vessel were reinforced with muslin wrapping. The postoperative course was uneventful. Seven years later, the patient has no neurological symptoms except for mild diplopia.

Case 6

This 27-year-old woman had a sudden attack of weakness on the right side after several months of intermittent posterior cervical and suboccipital head-
aches, and was hospitalized for further evaluation. Neuro-
ological assessment revealed bilateral ptosis, multi-
directional nystagmus, dysphagia, dysarthria, and ataxia on the left side. The patient had no history of illness, but her blood pressure measured routinely on two occasions 7 months before had been 160/110 and 140/85 mm Hg, and she had taken oral contraceptive agents for several years. A CT scan was normal and lumbar puncture revealed no blood in the CSF. An-
giography showed an irregular narrowing of the left vertebral artery, beginning above the C-2 vertebral level and extending to the basilar junction. These findings
were consistent with a dissecting aneurysm of the vertebral artery that began extracranially. The patient's symptoms gradually abated and she was discharged to a rehabilitation facility. Four years later, the patient is independent, although she has a persistent right hemiparesis and horizontal nystagmus.

Review of the Literature

We reviewed the world literature from 1924 to 1983, and found 36 previous cases of intracranial dissecting aneurysms of the posterior circulation (Table 1). The patients were 15 to 69 years old (mean 35.8 years), and males outnumbered females by a ratio of 2.6 to 1. In only seven patients was hypertension documented before the onset of symptoms. Two women had used oral contraceptives. Trauma was not a direct cause in any case, and 60% of the cases reported before 1950 were attributed to syphilitic arteritis. Severe headache, primarily of the suboccipital region, was the most common prodromal and presenting symptom (72%). The clinical signs of an arterial dissection involving the posterior cerebral circulation invariably reflected focal or diffuse brain-stem ischemia.

The angiographic findings were diverse, ranging from complete occlusion to irregular narrowing of variable extent with or without an accompanying dilatation. The sole pathognomonic sign, a double lumen on angiography, was reported only once.4 A few patients had CT scans, but the findings were not helpful in diagnosing an acute arterial dissection. Lumbar puncture was performed in 27 patients and was unequivocal for SAH in seven. In all patients with SAH, a dissection between the media and adventitia was documented; three patients with the same pathological finding did not have SAH. Dissection between the internal elastic lamina and media was the most common location for the intramural hematoma. Thirty (83%) of these 36 patients died within a few days to weeks after their presentation; among the six survivors, one patient had surgical exploration only, four patients underwent a definitive procedure to exclude, partially or completely, the dissecting segment from the remaining circulation, and one patient was treated conservatively. Anticoagulant therapy was not used in any case.

Discussion

Dissecting aneurysms of the posterior circulation are an uncommon but distinct clinical entity that must be distinguished from dissections of the systemic and cervical arteries. Differences in the etiology, clinical and diagnostic findings, and treatment are discussed in the following sections.

Pathogenesis

Pathologically, there are important differences between dissecting aneurysms of the extracranial arteries and those of the intracranial vessels. In the extracranial arteries, dissection occurs in the outer layers of the media or between the media and adventitia.22,44,70,85 Aortic dissections, for example, result from accumulation of acid mucopolysaccharides and cystic degeneration of the media,2,14 which subsequently cause rupture of the vasa vasorum28 and formation of an intramural hemorrhage. Dissection in other systemic (for instance, renal and coronary) arteries is rare,22 but also occurs between the media and adventitia.82 Deposition of mucoid material has been known to involve these layers, and medial degeneration has been identified in nearly 25% of systemic dissections.31 Both of these pathological conditions have been attributed to an aging phenomenon.6 Dissections of the cervical carotid and vertebral arteries also occur in the outer layers of the media, or between the media and adventitia.6,56,63 Cystic medial necrosis and degeneration of elastic tissue in the media have been observed in extracranial carotid dissections.3,34,54 An isolated intimal defect in the cervical carotid artery is an uncommon finding,26,46 and would support either a primary structural or biochemical defect of the media, or a secondary cause, such as a toxic insult or rupture of a reactive neovascular network formed from a change in the vasa vasorum.79

In nearly all intracranial arterial dissections, the intramural hematoma forms between the internal elastic lamina and the media.1,4,44,50,51,85,86 Rarely, the dissection involves the subadventitia and may rupture into the subarachnoid space.2,39,66,72,81,86 It is not clear why the location of the intramural hematoma in intracranial arterial dissections is different from that in extracranial dissections. However, anatomical differences in these two vascular systems may be involved.

The intracranial arteries lack an external elastic membrane and have a thinner adventitia, fewer elastic fibers in the media, and, generally, a thicker internal elastic lamina than the extracranial arteries.20,70 In the cervical carotid arteries, these changes occur at the level of the skull base. In the vertebral artery, they begin 1 cm proximal to its dural perforation and, within 6 mm of that point, elastic fibers in the adventitia and media are absent.84 Systemic vascular dissections may be attributed to a defect in the media followed by rupture of the vasa vasorum; but, for two reasons, this explanation does not hold true for dissections of the intracranial arteries. First, the media and internal layer of the intracranial vessels lack vasa vasorum.5 Minute vessels have been identified in areas of focal intimal hyperplasia34 and in atherosclerotic plaques,59 but these pathological features are rarely seen with an intracranial dissection.24,52,73 Second, medial defects are common25,75 and have been found in up to 75% of cerebral arteries from normal brains.29 Although these defects are not often identified in the first 10 years of life, they become more common with age,13,20 and intracranial dissection is too rare for there to be a significant relationship. Stehbens75 examined nearly 300 human arterial bifurcations and found many medial defects in the anterior cerebral circulation, few at the
### TABLE 1

*Summary of 36 previously reported cases of vertebrobasilar dissection*

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age (yrs), Sex</th>
<th>Involved Vessel(s)</th>
<th>Medical History</th>
<th>Presenting Symptoms &amp; Signs</th>
<th>CSF</th>
<th>CT</th>
<th>Angiography</th>
<th>Surgery</th>
<th>Outcome</th>
<th>Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schoefield, 1924</td>
<td>47, M</td>
<td>rt vertebral, basilar</td>
<td>syphilis(?)</td>
<td>cervical pain, headaches, diplopia, nystagmus, dysphagia, facial droop, dysmetria of the upper extremities, stupor, abrupt coma</td>
<td>clear</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>death in 15 days</td>
<td>dissection of vertebrobasilar “wall” causing occlusion</td>
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<tr>
<td>Hyland, 1933</td>
<td>42, M</td>
<td>rt vertebral, basilar</td>
<td>neurosyphilis</td>
<td>headaches (intense), rt hemiparesis, facial weakness, diplopia, rt hemiparesis</td>
<td>bloody</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>death in 12 days</td>
<td>mucoid degeneration of media; dissection: media/adventitia</td>
</tr>
<tr>
<td>Stern, 1933</td>
<td>24, M</td>
<td>basilar, PCA, AChA</td>
<td>syphilis</td>
<td>“heat stroke,” abrupt coma</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>death in 12 days</td>
<td>syphilitic aortitis, medial arteritis, intramural dissecting hematoma</td>
</tr>
<tr>
<td>Hasain, 1937</td>
<td>37, M</td>
<td>basilar</td>
<td>accidental electrocution</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>death in 6 days</td>
<td>disruption of media, questionable atherosclerosis; dissection: intima/media</td>
<td></td>
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<tr>
<td>Szabo, 1939</td>
<td>35, F</td>
<td>rt vertebral</td>
<td>syphilitic arteritis</td>
<td>“signs of an intracranial tumor,” headache × 4 weeks</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>trephine: negative</td>
<td>death in 12 days</td>
<td>syphilitic arteritis, medial arteritis, intramural dissecting hematoma</td>
</tr>
<tr>
<td>de Busscher, 1952</td>
<td>45, M</td>
<td>rt vertebral, basilar</td>
<td>negative</td>
<td>“cerebellopontine angle syndrome”</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>trephine: negative</td>
<td>death in 5 days</td>
<td>disruption of media, questionable atherosclerosis; dissection: intima/media</td>
</tr>
<tr>
<td>Watson, 1956</td>
<td>32, M</td>
<td>basilar</td>
<td>negative</td>
<td>suboccipital headaches, cervical tenderness, confusion, lethargy</td>
<td>clear</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>death in 4 days</td>
<td>cystic medial degeneration, adventitia &amp; intima intact, brain-stem infarct; dissection: intima/media</td>
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<tr>
<td>Krayenbühl, 1957</td>
<td>55, F</td>
<td>PICA</td>
<td>INA</td>
<td>“suspected aneurysmal rupture,” coma</td>
<td>INA</td>
<td>INA</td>
<td>—</td>
<td>—</td>
<td>death in 5 days</td>
<td>INA</td>
</tr>
<tr>
<td>Wolman, 1959</td>
<td>33, F</td>
<td>basilar, rt PCA, SCA</td>
<td>negative</td>
<td>headaches × 1 week, abrupt coma, extensor posturing</td>
<td>clear</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>death in 7 days</td>
<td>brain-stem infarct, “congenital mural defect;” dissection: intima/media</td>
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<tr>
<td>Crosato &amp; Terzian, 1961</td>
<td>30, M</td>
<td>basilar, rt PCA, lt SCA</td>
<td>negative</td>
<td>recurrent headache, abrupt coma</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>death in 5 days</td>
<td>signs of atheroma; dissection: intima/ media</td>
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<tr>
<td>Perrier, <em>et al.</em>, 1964</td>
<td>22, F</td>
<td>basilar</td>
<td>post-partum 15 days</td>
<td>headaches, lt hemiparesis, facial weakness, lt 3rd nerve palsy, coma</td>
<td>clear</td>
<td>—</td>
<td>carotid only: negative</td>
<td>—</td>
<td>death in 30 days</td>
<td>disruption of IEL &amp; media; dissection: intima/media</td>
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<tr>
<td>Perrier, <em>et al.</em>, 1966</td>
<td>21, M</td>
<td>basilar, lt PCA</td>
<td>negative</td>
<td>headaches, nystagmus, coma, flaccid quadriplegia</td>
<td>clear</td>
<td>—</td>
<td>carotid only: normal</td>
<td>—</td>
<td>death in 16 days</td>
<td>disruption of IEL &amp; media; dissection: intima/media (these defects also seen in extracranial vessels)</td>
</tr>
<tr>
<td>Hayman &amp; Anderson, 1966</td>
<td>15, M</td>
<td>basilar</td>
<td>mental retardation</td>
<td>abrupt coma, extensor posturing on lt, flaccid ca rt</td>
<td>clear</td>
<td>—</td>
<td>bilat carotid: negative</td>
<td>—</td>
<td>death in 8 wks</td>
<td>medias disruption; dissection: media</td>
</tr>
<tr>
<td>Redondo-Marco &amp; Walb, 1967</td>
<td>30, F</td>
<td>rt vertebral, basilar</td>
<td>concussion</td>
<td>4-wk headache, abrupt coma, posturing</td>
<td>clear</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>death in 7 days</td>
<td>“congenital defect,” IEL in many layers, collagen in intima; dissection: intima/media, adventitia &amp; media normal</td>
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<tr>
<td>Author</td>
<td>Case No</td>
<td>Side</td>
<td>Artery</td>
<td>Location</td>
<td>Symptoms</td>
<td>CT Findings</td>
<td>Follow-up</td>
<td>Comment</td>
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<td>Brihaye, et al., 1971</td>
<td>22, M</td>
<td>basilar</td>
<td>automobile accident 10 yrs before</td>
<td>headaches, abrupt coma, flaccid; then became lethargic with residual facial weakness &amp; EOM palsy</td>
<td>clear</td>
<td>vert: basilar occlusion</td>
<td>death in 2 mos</td>
<td>defective IEL; dissection: intima/media</td>
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<tr>
<td>Nožička, 1972</td>
<td>40, M</td>
<td>lt vertebral</td>
<td>INA</td>
<td>headaches, N/V, coma, abnormal respirations</td>
<td>clear</td>
<td>vert: basilar occlusion</td>
<td>death, 7 time</td>
<td>dissection: intima/media</td>
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<td>Pesendorfer &amp; Platthy, 1973</td>
<td>35, F</td>
<td>basilar</td>
<td>oral contraceptives, long history of headaches</td>
<td>abrupt coma, flaccid</td>
<td>INA</td>
<td>INA</td>
<td>death in 48 hrs</td>
<td>mucopolysacchar in vessel wall, IEL abnormal; dissection: intima/media</td>
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<tr>
<td>Escourrolle, et al., 1973</td>
<td>32, M</td>
<td>basilar, rt vertebral</td>
<td>questional recent trauma</td>
<td>occipital headaches, coma, flaccid</td>
<td>clear</td>
<td>vert: basilar occlusion</td>
<td>death in 5 days</td>
<td>IEL &amp; intima fragmented, media normal; dissection: intima/media</td>
<td></td>
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<tr>
<td>Dourov &amp; Flament-Durand, 1975</td>
<td>22, M</td>
<td>distal basilar, lt PCA, lt SCA</td>
<td>negative</td>
<td>headaches, abrupt coma, nystagmus, extensor posturing severe</td>
<td>clear</td>
<td>vert: basilar occlusion</td>
<td>death in 58 hrs</td>
<td>IEL disrupted &amp; duplicated; dissection: intima/media</td>
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<tr>
<td>Pasquier, et al., 1976</td>
<td>32, M</td>
<td>basilar, rt PCA, rt vertebral</td>
<td>negative</td>
<td>headaches severe, hemiplegia, rt 7th nerve palsy, locked-in syndrome</td>
<td>clear</td>
<td>narrowing of rt vertebral lt vertebral normal; basilar occlusion</td>
<td>death in 2 mos</td>
<td>dissection began immed. proximal to dural penetration of rt vert;</td>
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<td>subintimal fibrosis seen in dissection;</td>
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<td>dissection: intima/media</td>
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<tr>
<td>Yonas, et al., 1977</td>
<td>44, F</td>
<td>rt vertebral</td>
<td>INA</td>
<td>intense occipital headaches rt &gt; lt, photophobia, stiff neck</td>
<td>bloody</td>
<td>fusiform dilation of vertebral starting at PICA origin; segmental narrowing in middle of dilated segment</td>
<td>vertebal ligation above PICA origin</td>
<td>death in 3 days postop</td>
<td>dissection began immed. proximal to dural penetration of rt vert;</td>
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<td>subintimal fibrosis seen in dissection;</td>
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<td>dissection: intima/media</td>
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<td></td>
</tr>
<tr>
<td>Waga, et al., 1978</td>
<td>53, M</td>
<td>rt vertebral</td>
<td>INA</td>
<td>severe headache, stiff neck, subhyloid hemorrhage</td>
<td>bloody</td>
<td>fusiform dilation surrounded by dark red clot; PICA distal to dilated segment; clipped prox vertebral intracranially</td>
<td>alive</td>
<td>dissection: adventitia/media (at exploration)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fisher, et al., 1978</td>
<td>18, M</td>
<td>rt PCA</td>
<td>INA</td>
<td>abrupt rt neck pain, lt side numbness, lt homonymous hemianopsia</td>
<td>clear</td>
<td>narrowed rt P2 segment extending distally 2.5 cm; repeat angiogram 3 mos later: no change</td>
<td>alive</td>
<td>dissection: adventitia/media</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alexander, et al., 1979</td>
<td>69, M</td>
<td>prox basilar &amp; terminal status, hypertension</td>
<td>altered mental status, incontinence, paraparesis, N/V, xanthochromic, increased reflexes</td>
<td>1.5 x 3 cm enhancing mass anterior to brain stem</td>
<td>xanthochromic mass anterior to brain stem</td>
<td>exploration of fibrous walled mass (basilar) filled with clotted blood</td>
<td>death in 3 days postop</td>
<td>arteriosclerosis; 1 cm from basilar origin, expanded into brown mass incorporating terminal vertebral segments, SAH verified; dissection: adventitia/media</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>expanded distal basilar, pontine perforators &amp; SCA; dissection: intima/media</td>
<td></td>
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<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>dissection: intima/media</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pasquier, et al., 1979</td>
<td>43, F</td>
<td>lt vertebral, lt PICA, prox basilar</td>
<td>mild hypertension, mild trauma 2 yrs before</td>
<td>abrupt coma, flaccid</td>
<td>clear</td>
<td>negative</td>
<td>death in 14 days</td>
<td>fibrodysplasia of intima in renal arteries also; dissection: intima/media/ adventitia</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*INA = information not available; — = not done; AChA = anterior choroidal artery; PCA = posterior cerebral artery; PICA = posterior inferior cerebellar artery; SCA = superior cerebellar artery; SAH = subarachnoid hemorrhage; IEL = internal elastic lamina; EOM = extraocular muscle; N/V = nausea/vomiting; CSF = cerebrospinal fluid; CT = computerized tomography.

Table 1 (continued)
<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age (yrs), Sex</th>
<th>Involved Vessel(s)</th>
<th>Medical History</th>
<th>Presenting Symptoms &amp; Signs</th>
<th>CSF</th>
<th>CT</th>
<th>Angiography</th>
<th>Surgery</th>
<th>Outcome</th>
<th>Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Takita, et al., 1979</td>
<td>33, M</td>
<td>basilar, rt vertebral</td>
<td>hypertension × 4 yrs</td>
<td>headache, N/V, lt side weakness, vertigo, × 1 hr then resolved; 24 hrs later severe headache; 4 mos later lt hemiparesis, stupor; 48 hrs later coma</td>
<td>1st clear</td>
<td>2nd bloody</td>
<td>vert: dilation of both terminal vertebral &amp; occlusion lt PCA</td>
<td>__</td>
<td>death in 4 days after 2nd ictus</td>
<td>defect of IEL dissection: intima/media/adventitia</td>
</tr>
<tr>
<td>Martini, 1979</td>
<td>47, M</td>
<td>vertebral: lt &gt; rt, basilar, rt PICA</td>
<td>vertigo 3 wks before admission, occipital headache</td>
<td>abrupt coma, flaccid</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>death in 6 days</td>
<td>IEL fragmented, media thin &amp; partly absent, intima fibrotic; dissection: intima/media</td>
</tr>
<tr>
<td>Sekino, et al., 1982</td>
<td>44, M</td>
<td>rt vertebral, basilar</td>
<td>hypertension (treated)</td>
<td>lt side numbness, followed by abrupt motor weakness on lt including face</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>alive, stable</td>
<td>dissection: adventitia/media (at exploration)</td>
</tr>
<tr>
<td>Senter &amp; Sarwar, 1982</td>
<td>45, M</td>
<td>lt vertebral</td>
<td>negative</td>
<td>headaches, cervical pain, ataxia × 24 hrs; patient previously had lower brain-stem ischemia that resolved 3 wks later</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>alive, stable</td>
<td>__</td>
</tr>
<tr>
<td>Adams, et al., 1982</td>
<td>39, F</td>
<td>basilar</td>
<td>negative</td>
<td>abrupt headaches, bilat 6th nerve paresis, lt facial weakness, stiff neck, rt hemiparesis, coma 4 days later</td>
<td>bloody</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>death in 5 days</td>
<td>SAH; atherosclerotic narrowing of basilar; IEL &amp; media: defects &amp; disrupted; dissection started at IEL → media → adventitia</td>
</tr>
<tr>
<td>Kulla, et al., 1982</td>
<td>37, M</td>
<td>vertebral lt &amp; rt, basilar, prox rt PCA</td>
<td>polycystic kidney disease, hypertension</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>death in 69 days</td>
<td>dissection: adventitia/media in rt &amp; lt vertebral basilar, prox rt PCA; IEL &amp; intima intact</td>
<td></td>
</tr>
<tr>
<td>Bugiani, et al., 1983</td>
<td>31, F</td>
<td>mid basilar</td>
<td>smoking 15 yrs, oral contraceptives 1 yr, hypertension 1 yr</td>
<td>postcervical headaches; 10 days before admission: ataxia, dysphagia, nystagmus, diplopia, facial hypotension; then locked-in syndrome</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>death in 16 days</td>
<td>dissection: intima/media; pons infarct</td>
</tr>
<tr>
<td>Kalyan-Raman, et al., 1983</td>
<td>38, M</td>
<td>rt SCA</td>
<td>negative</td>
<td>severe headaches, vertigo, diplopia, lethargy</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>death in 48 hrs</td>
<td>fibromuscular displasia, IEL disrupted; transp dissection: intima/media dissection: adventitia/media; IEL defect opened, media/adventitial dissection</td>
</tr>
<tr>
<td>Berkovic, et al., 1983</td>
<td>40, M</td>
<td>basilar</td>
<td>borderline hypertension</td>
<td>abrupt coma, flaccid</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>__</td>
<td>death in 15 days</td>
<td>__</td>
</tr>
</tbody>
</table>

* INA = information not available; — = not done; AChA = anterior choroidal artery; PCA = posterior cerebral artery; PICA = posterior inferior cerebellar artery; SCA = superior cerebellar artery; SAH = subarachnoid hemorrhage; IEL = internal elastic lamina; EOM = extraocular muscle; N/V = nausea/vomiting; CSF = cerebrospinal fluid; CT = computerized tomography.
Dissecting aneurysms of the posterior circulation

vertebrobasilar junction, and none at the basilar bifurcation.

Glynn\textsuperscript{26} found that cerebral arteries \textit{in vitro} could support a pressure twice as great as the highest recorded blood pressure \textit{in vivo}. He concluded that the internal elastic lamina, which contains the elastic tissue in the intracranial arteries, was the most important layer for determining the strength of the vessel wall. Even in the absence of an atheroma, the vessel was more prone to damage if the elastic tissue was either deficient or defective. Some investigators believe that intimal cushions, which are formed at branch points in the cerebral circulation by splitting of the internal elastic lamina, may serve as a starting point for dissection, especially if an underlying medial defect is present.\textsuperscript{33,35} Others believe that a defective elastic layer is the origin of an intramural dissection,\textsuperscript{16,24,36,40,70} and that the true lumen is the source of the intramural hemorrhage. Whether the defect in the elastic layer is congenital or acquired is not known. In rare instances, the dissection occurs between the media and adventitia, which usually results in SAH. Although the initial event may resemble an incident that precipitates a dissection between the internal elastic layer and the media, subsequent rupture into the subadventitial plane and subarachnoid space may occur.\textsuperscript{1} This may be more likely in older persons, who have more defects in the vessel wall and a thinner adventitia. Among patients with vertebrobasilar dissections, those who suffered SAH were generally older than those who did not.

Oral contraceptive agents have been widely implicated in the pathogenesis of thromboembolic disease and cerebral infarction.\textsuperscript{29} Studies in human arteries have shown that birth control pills can cause structural alterations, including endothelial proliferation and intimal thickening.\textsuperscript{69} Two patients reported elsewhere and two of our own patients were taking oral contraceptives before the onset of their symptoms.

\textbf{Clinical Features}

Intracranial arterial dissections occur in young adults (aged in the late 20's through early 40's). The patients are rarely hypertensive and few have a history of atherosclerotic peripheral vascular disease or diabetes.\textsuperscript{4,24,30,44,52} In contrast, patients with dissections of the aorta or other systemic arteries are older (50 to 70 years old) and generally have hypertension and arteriosclerosis.\textsuperscript{22,31} Spontaneous cervical carotid dissections are often associated with hypertension,\textsuperscript{18,54} and may be associated with cystic medial necrosis\textsuperscript{10,56,79} and fibromuscular dysplasia.\textsuperscript{18} Cervical dissections tend to occur in young adults of the same age group as those with intracranial dissections. The majority of dissecting intracranial aneurysms have no recognized cause,\textsuperscript{56} although isolated examples of intracranial dissections associated with syphilis,\textsuperscript{39,77,80} migraine,\textsuperscript{2,73} cystic necrosis of the media,\textsuperscript{59} fibromuscular dysplasia,\textsuperscript{3} homocystinuria,\textsuperscript{12} and trauma have been reported.

The most common symptom of vertebrobasilar dissection before the onset of brain-stem dysfunction is headache.\textsuperscript{11,86} Including our six patients, 76% of all reported patients with vertebrobasilar dissections had headaches. The pain was usually in the occipital and posterior cervical regions and intensified immediately before clinical deterioration. No other pathognomonic symptom or sign of vertebrobasilar dissecting aneurysm has been identified.

\textbf{Angiographic Features}

Although no single finding has been present in all cases, the angiographic features in patients with intracranial dissecting aneurysms\textsuperscript{72,81} enable a diagnosis to be made in conjunction with the clinical presentation.\textsuperscript{11,30,47} A narrow tapered lumen ("string sign"), a common feature of cervical carotid and vertebral dissections,\textsuperscript{7} is often observed with cerebral dissections.\textsuperscript{44} The lumen is usually irregular and asymmetrical and is often severely stenotic. Yet a narrow tapered lumen is often seen in conjunction with a proximally dilated segment or an irregular poststenotic enlargement ("string and pearl sign").\textsuperscript{68,86} If the intramural clot becomes large enough, a complete occlusion follows.\textsuperscript{45} A double lumen, representing the passage of contrast material into both true and false channels, is a pathognomonic finding of intracranial dissection,\textsuperscript{4,23,81} but is extremely rare.\textsuperscript{35} In two of our cases, a change in the luminal configuration over several weeks supported the diagnosis, retrospectively, of a dissecting aneurysm. This evolving angiographic feature may serve as a complementary diagnostic sign, but it has been reported only once before in a patient with vertebrobasilar dissection who underwent repeat angiography.\textsuperscript{1}

\textbf{Treatment}

In 1951, Poppen\textsuperscript{64} reported two patients with middle cerebral artery dissection in whom surgical treatment halted progression of the deficits. This was the first attempt at surgical intervention for intracranial dissecting aneurysms. Since then, very few patients have undergone a definitive surgical procedure for similar lesions, especially those in the posterior fossa. Waga, \textit{et al.},\textsuperscript{81} and Senter and Sarwar\textsuperscript{72} clipped the proximal vertebral artery intracranially in a patient with a vertebral dissection that ended proximal to the PICA origin. Yonas, \textit{et al.},\textsuperscript{86} ligated the vertebral artery distal to the PICA origin where the dissection began. In a case in which the dissection involved both the vertebral and basilar arteries, Sekino, \textit{et al.},\textsuperscript{71} trapped the vertebral artery above its dural entrance and distally, proximal to the origin of the PICA. One of these four patients died from unrelated causes, and the other three survived without incurring further neurological dysfunction.

Five of our six patients underwent an operative procedure: exploration of vertebral dissection in one, trapping of vertebral dissection in one, excision of the distal PCA dissection in one, and reinforcement of a
distal basilar dissection with muslin in two. Postoperatively, the patient who had a trapping procedure suffered a cerebellar and brain-stem infarction. This patient also had ventricular enlargement, as did the one who underwent exploration only. The patient whose dissection was excised suffered a mild hemiparesis and a transient movement disorder. The remaining two patients had an eventful postoperative course.

Anticoagulation therapy for intracranial dissections has not been widely advocated because of the potential for subsequent rupture, intracerebral hematoma, and extension of preexisting dissection. In contrast, most spontaneous dissections of the cervical carotid arteries resolve without treatment, although anticoagulation therapy is advisable to prevent ischemic complications. However, if recurrent transient ischemic events occur, or if a progressive deficit develops, surgical intervention becomes necessary. Extracranial vertebral artery dissections, which are much less common than vertebrobasilar dissections, also resolve spontaneously and should be treated with anticoagulant therapy. Vertebral ligation may be an alternative initial treatment.

We recommend exploration of vertebrobasilar dissecting aneurysms in patients with or without SAH who are clinically stable. Vertebral artery dissections proximal to the origin of the PICA should be trapped intracranially to prevent collateral flow from cervical muscular branches; if the dissection originates extradurally, a high cervical ligation can be performed as part of the trapping procedure. Temporary occlusion with an inflatable balloon may identify patients who would benefit from an extracranial-PICA bypass before trapping. A dissecting aneurysm that originates distal to the PICA origin and does not involve the vertebrobasilar junction may also be excluded from the posterior circulation by a high cervical ligation. The goal of an operative procedure for basilar artery dissection would be to reinforce (wrap) as much of the abnormal vessel as possible. Although this may seem unjustified, reinforcement may be particularly beneficial in patients with SAH. Dissecting aneurysms of the distal branches of the vertebrobasilar system can be treated by excision, supplemented with a bypass procedure if the lesion is proximally located.

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Dissecting aneurysms of the posterior circulation

due to spontaneous dissection of the internal carotid artery. Arch Neurrol 40:448-449, 1983


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